**Conclusion:** This case emphasizes the potential for stone formation when non-absorbable sutures are used in the urinary tract. When we encounter a urolithiasis that locates over the place where a prior surgical procedure was done, we should keep in mind to find out any possible non-absorbable material remains.

**PD3-2:**

**TRPV1 HYPERFUNCTION IMPAIRS RENAL SENSORY RESPONSE IN THE RAT HYPEROXALURIC KIDNEY**

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**Purpose:** We previously demonstrated that renal sensory function in response to mechano- and chemo-stimulation was impaired in rat kidneys with hyperoxaluria and/or calcium oxalate (CaOx) crystals. The transient receptor potential vanilloid 1 channel (TRPV1) is known to present in rat kidneys and responsible for activation of afferent renal nerve activity (ARNA) and induction of diuretic renorenal reflex. The present study therefore tests whether TRPV1 dysfunction may be the underlying mechanism for contribution to sensory impairment seen in the hyperoxaluric kidney.
**Materials and Methods:** Acute hyperoxaluria was induced by intrapelvic perfusion of oxalate into renal pelvis, a tissue area mostly originated for renal sensory nerves. Chronic hyperoxaluria was induced in rats after fed with fed with 5% hydroxyl-L-proline (HP) in daily diet for 42 days. Renal nerves in the left kidney were carefully isolated for recording of ARNA and efferent renal sympathetic nerve activity (ERSNA). Changes in the cortical microvascular blood flow (CMBF) were monitored by ultrasonic flowmeter. Renorenal reflex response was tested by rising intrapelvic pressure (IPP) or intrapelvic perfusion of high salt solution in the left kidney, and urine output and sodium excretion in the contralateral (right) kidney were collected. Renal pelvic effluent was collected for determination of the amount of sensory neuropeptide substance P (SP) release. Changes in TRPV1 and neurokinin-1 receptor (NK-1R) expression in renal pelvis were examined by Western blot analysis.
**Results:** Compared to vehicle-treated kidney, acute perfusion of oxalate into renal pelvis resulted in persistently decreased ARNA, increased ERSNA, decreased CMBF, and impaired renorenal reflex after 4 h of treatment. SP release was increased after oxalate treatment, this associated with NK-1R downregulation. These however were attenuated by co-treatment of specific TRPV1 blocker SB-366791. As a polymodal sensor, our results indicate TRPV1 may act as an oxalate sensor. In HP rats, ARNA in response to chemo- and mechano-stimulation, and ARNA-mediated reflex control on ERSNA and renal excretion were impaired after 7 days of treatment and thereafter. Severe reductions were found after 42 days of hyperoxaluria with CaOx deposition. These associated with increases in TRPV1 expression and SP release, and NK-1R downregulation in renal pelvis.
**Conclusion:** Our results clearly indicate that TRPV1 hyperfunction contributes to renal sensory impairment in the hyperoxaluric rats, which led to too much SP release, and NK-1R desensitization in nerve endings of renal pelvis. Deficiency in renal sensation blinds the kidney to the presence of hyperoxaluria as well as the formation of CaOx.

**PD3-3:**

**MOBILIZATION OF ENDOGENOUS STEM CELLS ATTENUATES TUBULAR CELL DAMAGE AND CALCIUM OXALATE CRYSTAL FORMATION IN THE RAT HYPEROXALURIC KIDNEY**

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**Purpose:** Cell therapy has been demonstrated to be beneficial for treatment of disease by enhancing self and/or endogenous repair in various organ tissues. Whether cell therapy is effective to alleviate tubular cell injury caused by hyperoxaluria during kidney stone formation is still unclear. This study aims to test whether mobilization of endogenous stem cells from bone marrow may protect kidneys against hyperoxaluria and further calcium oxalate (CaOx) crystal deposition.
**Materials and Methods:** Male Wistar rats were divided into control rats received standard diet and hyperoxaluric rats fed with 5% hydroxyl-L-proline (HP) in daily diet for 7 and 28 days. Another group of HP rats was received plerixafor, a selective CXCR4 blocker (AMD3100), continuously via a subcutaneous implant of mini-osmotic pump as CXCR4 antagonism is known to mobilize endogenous stem/progenitor cells into circulation. The 24 h of urine sample was collected to evaluate degree of supersaturation by estimation of the ionic activity of calcium oxalate (CaOx) and the amount of calcium crystal excretion in urine sedimentation. Blood sample was collected to evaluate the circulating level of $CD34^+CXCR4^+$ cells. Crystal distribution within the kidneys will be examined by counting crystal deposits by von Kossa staining. Protein expressions in renal tissues and in urine were quantitatively analyzed.
**Results:** Compared to the vehicle-treated HP rats, plerixafor significantly attenuated CaOx crystal deposition and increased the amount of urinary sediment after 28-day treatment but without any effect on hyperoxaluria and supersaturation at both time-points. Interestingly, circulating $CD34^+CXCR4^+$ cells were markedly elevated in the plerixafor-treated rats after 7 days and persisted thereafter. This was associated with an increase in renal expression of stromal cell-derived factor 1 (SDF-1) in the plerixafor-treated kidneys for 7 and 28 days. The urinary contents of two anti-crystallization molecules, osteopontin (OPN) and Tamm-Horsfall protein (THP), in the plerixafor-treated HP rats were significantly increased as compared to those in the vehicle-treated HP groups for 7 and 28 days. This associated with an attenuation of enzymuria for tubular damage markers, α- and μ-glutathione-S-transferase (GST). Moreover, renal contents of OPN, THP, αGST, and μGST in the plerixafor-treated HP rats were higher than those in the vehicle-treated HP groups at both time-points.
**Conclusion:** These results clearly indicate that the anticrystallization effect of plerixafor is possibly related to an increase in $CD34^+CXCR4^+$ cell homing to the injured hyperoxaluric kidney with a higher SDF-1 expression, which attenuates tubular cell injury and against CaOx crystal formation by maintenance of renal production of OPN and THP. Attenuation of cell debris desquamated from damaged tubular cells may prevent seeding effect for calcium crystal formation in growth.

**PD3-4:**

**LONG-TERM PRESCRIPTION OF α-BLOCKERS DECREASE THE RISK OF RECURRENT UROLITHIASIS NEEDED FOR SURGICAL INTERVENTION-A NATIONWIDE POPULATION-BASED STUDY**

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**Purpose:** α1 receptors and subtypes have been confirmed to distribute in human pelvis and calyces recently. As used in ureteral stones, α-blocker treatment may facilitate kidney stone passage and long-term prescription of α-blocker may decrease the risk of recurrent urolithiasis. The aim of this study is to determine if use of α-blockers 180 days or more can decrease the risk of recurrent urolithiasis needed for surgical intervention.
**Materials and Methods:** A representative database of 1,000,000 patients from Taiwan's National Health Insurance was analyzed. Eligible patients were those who had received the first-time procedure for upper urinary stone removal, including extracorporeal shock-wave lithotripsy, ureterorenoscopic lithotripsy, or both, between 2000 and 2010. After completing a 180-day treatment for first event, patients were prospectively followed-up until a second set of stone procedures was performed (proxy of stone recurrence), loss to follow-up, or end of study. The effect of percentage of total number of days of α-blocker use on need for second set of stone